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FILE 'REGISTRY' ENTERED AT 15:25:46 ON 14 MAY 2002

=> S DUAL SPECIFICITY PHOSPHATASE/CN
L1             0 DUAL SPECIFICITY PHOSPHATASE/CN

=> S DUAL SPECIFICITY PHOSPHATASE
           126 DUAL
           215 SPECIFICITY
         10067 PHOSPHATASE
             6 PHOSPHATASES
         10067 PHOSPHATASE
                 (PHOSPHATASE OR PHOSPHATASES)
L2            21 DUAL SPECIFICITY PHOSPHATASE
                 (DUAL(W)SPECIFICITY(W)PHOSPHATASE)

FILE 'CAPLUS' ENTERED AT 15:27:05 ON 14 MAY 2002

=> S DUAL SPECIFICITY PHOSPHATASE;S DSP4 OR (DSP(W)4);S HUMAN OR HOMO SAPIENS
         61965 DUAL
           171 DUALS
         62053 DUAL
                 (DUAL OR DUALS)
        146808 SPECIFICITY
         15273 SPECIFICITIES
        156364 SPECIFICITY
                 (SPECIFICITY OR SPECIFICITIES)
         97382 PHOSPHATASE
         21342 PHOSPHATASES
        103679 PHOSPHATASE
                 (PHOSPHATASE OR PHOSPHATASES)
L3            242 DUAL SPECIFICITY PHOSPHATASE
                 (DUAL(W)SPECIFICITY(W)PHOSPHATASE)


           222 DSP4
          1640 DSP
            75 DSPS
          1686 DSP
                 (DSP OR DSPS)
       4409327 4
           312 DSP(W)4
L4            475 DSP4 OR (DSP(W)4)


         997589 HUMAN
         293910 HUMANS
        1153663 HUMAN
                 (HUMAN OR HUMANS)
          21694 HOMO
            205 HOMOS
          21790 HOMO
                 (HOMO OR HOMOS)
            488 SAPIENS
            457 HOMO SAPIENS
                 (HOMO(W)SAPIENS)
L5         1153824 HUMAN OR HOMO SAPIENS

=> S L2,L3
             15 L2
L6           250 (L2 OR L3)

=> S (L4,L6) AND L5
L7           120 ((L4 OR L6)) AND L5

=> S L4 AND L5
L8            11 L4 AND L5

=> D 1-11 CBIB ABS

L8   ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2002 ACS
2001:629609   Document No. 135:340796  A growing family of dual specificity
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phosphatases with low molecular masses. Aoki, Naohito; Aoyama, Koji; Nagata, Miyuki; Matsuda, Tsukasa (Department of Applied Molecular Biosciences, Graduate School of Bioagricultural Sciences, Nagoya University, Nagoya, 464-8601, Japan). Journal of Biochemistry (Tokyo, Japan), 130(1), 133-140 (English) 2001. CODEN: JOBIAO. ISSN: 0021-924X. Publisher: Japanese Biochemical Society.

AB Five putative dual specificity protein phosphatases (DSPs), designated LMW-DSP1, - ***DSP4*** , -DSP6, -DSP10, and -DSP11, were cloned with a combination of RT-PCR and cDNA library screening strategies. Sequencing anal. revealed that all lacked the cdc25 homol. domain that is conserved in most known DSPs/MAP kinase phosphatases (MKPs). LMW-DSP1 exhibited the highest similarity to plant DSPs. LMW- ***DSP4*** exhibited the highest similarity to ***human*** YVH1 and rat GKAP, but its C-terminal region was much shorter than that of the ***human*** and rat clones. LMW-DSP6 was found to be identical to recently cloned TMDP, and LMW-DSP11 seemed to be a mouse ortholog of ***human*** VHR. LMW-DSP10 was found to have a DSP catalytic-like domain, but the crit. cysteine residue for catalytic activity was missing. Recombinant LMW-DSP1, -DSP6, and -DSP11 exhibited obvious and strong activity against an artificial low mol. substrate, para-nitrophenyl phosphate (pNPP). Recombinant LMW- ***DSP4*** exhibited slight but significant activity, whereas no activity was detected for LMW-DSP10. The phosphatase activity of the recombinant LMW-DSPs was inhibited by orthovanadate but not sodium fluoride. However, none of the DSPs could dephosphorylate MAP kinases such as ERK1, p38, and SAPK/JNK in transiently transfected COS7 cells under the conditions used. Northern blot anal. revealed that LMW-DSP1, -DSP6, -DSP10, and -DSP11 were specifically expressed in testis, while LMW- ***DSP4*** was broadly expressed. The testis-specific expression and apparent absence of dephosphorylation action on MAP kinases suggest that LMW-DSP1, -DSP6, -DSP10, and -DSP11 play specific roles in testis. Taken together, it is conceivable that a distinct class of low mol. mass DSPs is present and plays a role in dephosphorylating unknown mols. other than MAP kinases.

L8 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2002 ACS

2000:846817 Document No. 134:37427 Effects of noradrenaline depletion in the brain on response to novelty in isolation-reared rats. Lapiz, Maria Danet S.; Mateo, Yolanda; Parker, Terry; Marsden, Charles (School of Biomedical Sciences, Queen's Medical Centre, University of Nottingham Medical School, Nottingham, NG7 2UH, UK). Psychopharmacology (Berlin), 152(3), 312-320 (English) 2000. CODEN: PSCHDL. ISSN: 0033-3158. Publisher: Springer-Verlag.

AB Rationale: Social isolation from weaning in the rat produces a variety of neurochem. and behavioral effects in the adult that in part parallel changes seen in ***human*** schizophrenia. Objectives: The study investigated the effects of central noradrenaline (NA) depletion by the selective neurotoxin, N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine (***DSP*** - ***4***), on the behavior of isolation-reared rats. Methods: Male Lister hooded rats were reared singly or in groups after weaning. During week 2, the rats were tested in photocell activity cages and were then injected with ***DSP*** - ***4*** (25 mg/kg, IP). During week 4, rats were tested in the open field under the following conditions: open field alone, with two novel stimuli (T1), and with a familiar and a novel object (T2), and in the activity cages. Results: ***DSP*** - ***4*** significantly reduced cortical and hippocampal NA levels with no effect on the hypothalamus. Isolation-reared rats exhibited locomotor hyperactivity and reduced habituation to the testing arena, although their exploration of the novel objects in T1 was not significantly different from group-reared rats. ***DSP*** - ***4*** treatment in group-reared rats increased inner zone activity in the open field but did not significantly affect the exploration of novel objects. ***DSP*** - ***4*** treatment in isolates reduced exploration of objects at T2 while increasing exploration of the general environment. Conclusions: Isolation rearing influences the behavioral effects of central NA depletion. The results suggest isolation-induced changes in the central noradrenergic system in the isolated rat, supporting the view that early environmental factors can have long-term effects on central noradrenergic function as well as other neurotransmitter systems.

L8 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2002 ACS

2000:725784 Document No. 133:306352 Protein and cDNA sequences of a novel

.***human*** protein ***DSP*** - ***4*** with dual-specificity MAP kinase phosphatase activity, and therapeutic uses thereof. Luche, Ralf M.; Wei, Bo (Ceptyr, Inc., USA). PCT Int. Appl. WO 2000060099 A1 20001012, 63 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US9313 20000407. PRIORITY: US 1999-PV128204 19990407.

AB The invention provides protein and cDNA sequences of a novel ***human*** protein ***DSP*** - ***4*** , which has sequences homol. with dual-specificity MAP kinase phosphatase. The protein ***DSP*** - ***4*** may be used, for example, to identify antibodies and other agents that inhibit ***DSP*** - ***4*** activity. North blotting results show significantly higher levels of ***DSP*** - ***4*** mRNA in tissues of ***human*** skeletal muscle and thymus. The invention further relates to the uses of protein ***DSP*** - ***4*** for modulating cell proliferation, differentiation and survival.

L8 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS

2000:135318 Document No. 133:37599 Neuroprotective and neuronal rescue effects of selegiline: review. Magyar, K.; Haberle, D. (Department of Pharmacodynamics, Semmelweis University of Medicine, Budapest, H-1089, Hung.). Neurobiology (Budapest), 7(2), 175-190 (English) 1999. CODEN: NROBEZ. ISSN: 1216-8068. Publisher: Akademiai Kiado.

AB A review with many refs. The effect of selegiline [(-)-deprenyl] cannot be considered as a simple, selective inhibitor of MAO-B. Pretreatment with the drug prevented the effect of specific neurotoxins like MPTP, 6-OH-dopamine, ***DSP*** - ***4*** and AF64A. Selegiline pretreatment prevented the depletion of noradrenaline (NA) induced by ***DSP*** - ***4*** in the rat hippocampus. This can be due to the uptake inhibitory effect of selegiline and mainly to its metabolite methylamphetamine (MA), which is more potent inhibitor of the re-uptake than the parent compd. SKF-525A pretreatment diminished the protective effect of selegiline against ***DSP*** - ***4*** , while phenobarbital pretreatment decreased its MAO-B inhibitory potency. Selegiline in low oral doses also prevented the effect of ***DSP*** - ***4*** due to its intensive "first pass" metab. Selegiline treatment can rescue damaged neurons. It inhibited the apoptosis in M-1 ***human*** melanoma cells in a rather low concn. (10-13M). The mode of action of the drug regarding the inhibition of apoptosis is not known.

L8 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2002 ACS

1999:707058 Document No. 132:30655 Differential effects of staurosporine and retinoic acid on the vulnerability of the SH-SY5Y neuroblastoma cells: involvement of Bcl-2 and p53 proteins. Tieu, K.; Zuo, D. M.; Yu, P. H. (Neuropsychiatry Research Unit, Department of Psychiatry, University of Saskatchewan, Saskatoon, SK, S7N 5E4, Can.). Journal of Neuroscience Research, 58(3), 426-435 (English) 1999. CODEN: JNREDK. ISSN: 0360-4012. Publisher: Wiley-Liss, Inc..

AB ***Human*** catecholaminergic neuroblastoma cells (SH-SY5Y) have been widely used in different neurochem. investigations. Quite often these cells are induced to differentiation by various agents, such as staurosporine and retinoic acid. Interestingly, even though both staurosporine and retinoic acid induce similar morphol. differentiation in SH-SY5Y cells, the authors found that these 2 groups of differentiated cells exhibited opposite vulnerability to harmful chems. and phys. insults. In the present study, cisplatin, 5-fluorouracil (5-FU), N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine (***DSP*** - ***4***), 6-hydroxydopamine (6-OHDA), and .gamma.-radiation were used to assess the tolerance of the differentiated cells. Cell viability was detd. by 3-(4,5-dimethylthiazol-2yl)-2,5-diphenyltetrazolium bromide (MTT) assay. Staurosporine-treated SH-SY5Y cells were more sensitive to these toxic insults than the untreated controls. In contrast, retinoic acid-treated cells became more resistant to the same treatments. The expression of the proteins of the protooncogene Bcl-2 and the tumor suppressor gene p53 following staurosporine or retinoic acid treatment was assessed by Western blot and immunocytochem. Retinoic acid increased Bcl-2 and decreased p53

levels, whereas staurosporine decreased Bcl-2 and increased p53 levels. The opposite alteration of Bcl-2 (anti-apoptotic) and p53 (apoptotic) contents in SH-SY5Y cells with retinoic acid and staurosporine are attributed to the changes in cell vulnerability. These observations also indicate that caution should be taken when chem. induced differentiated neuroblastoma cells are to be used as an in vitro model for studying neuronal survival.

L8 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2002 ACS
1998:330046 Document No. 129:103676 The neuroprotective and neuronal rescue effects of (-)-deprenyl. Magyar, K.; Szende, B.; Lengyel, J.; Tarczali, J.; Szatmary, I. (Department of Pharmacodynamics, Semmelweis University of Medicine, Budapest, Hung.). Journal of Neural Transmission, Supplement, 52(MAO - The Mother of all Amine Oxidases), 109-123 (English) 1998. CODEN: JNTSD4. ISSN: 0303-6995. Publisher: Springer-Verlag Wien.
AB A review with 41 refs. The pharmacol. effects of (-)-deprenyl is multi-fold in its nature (dopamine sparing activity, neuroprotective and neuronal rescue effects), which cannot be explained solely by the irreversible MAO-B inhibitory action of the substance. Deprenyl slightly inhibits the re-uptake of noradrenaline and dopamine, but methylamphetamine, the metabolite of the inhibitor, by one order of magnitude is more potent in this respect, than the parent compd. Neither the metabolite nor (-)-deprenyl acts on the uptake of serotonin. The inhibitor has an intensive first pass metab. after oral treatment. The in vivo pharmacokinetic studies with (-)-deprenyl, using the double labeled radioisotope technique (1.5 mg/kg; orally) in rats revealed that the molar concn. of methylamphetamine can reach the level suitable to induce a significant inhibition of amine uptake. Deprenyl, but esp. methylamphetamine pre-treatment can prevent the noradrenaline release induced by the noradrenergic neurotoxin ***DSP*** - ***4*** . The uptake inhibitory effect of (-)-deprenyl and the metabolites is reversible. After repeated administration of (-)-deprenyl (1.5 mg/kg daily, for 8 days) sustained concn. of its metabolites was detected, compared to that of the acute studies. This can at least partly explain why (-)deprenyl should be administered daily to evoke therapeutic effects in Parkinson's disease. Administration of (-)-deprenyl in a low dose, following the toxic insult, can rescue the damaged neurons. The neuronal rescue effect of the drug was studied on M-1 ***human*** melanoma cells in tissue culture. The inhibitor reduced the apoptosis of serum-deprived M-1 cells, but the (+)-isomer failed to exert this effect. The (.+-.)-desmethyl-deprenyl almost lacks the property to inhibit apoptosis. For neuroprotection and neuronal rescue an optimal dose of (-)-deprenyl should be administered, because to reach a well balanced concn. of the metabolites in tissues is crit.

L8 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2002 ACS
1997:659454 Document No. 127:314300 The pharmacology of B-type selective monoamine oxidase inhibitors; milestones in (-)-deprenyl research. Magyar, K.; Szende, B.; Lengyel, J.; Tekes, K. (Dep. Pharmacodynamics, Semmelweis Univ. Med., Budapest, Hung.). J. Neural Transm., Suppl., 48(Deprenyl--Past and Future), 29-43 (English) 1996. CODEN: JNTSD4. ISSN: 0303-6995. Publisher: Springer.
AB A review with .apprx.60 refs. (-)-Deprenyl cannot be considered as a simple, selective inhibitor of MAO-B. It increases the dopaminergic tone in the central nervous system by a complex mechanism. The MAO-B inhibition could result in a potentiation of the effect and the redn. of the dose of L-dopa, including the restoration of the sensitivity to L-dopa treatment, when the response to the drug has already been diminished or lost. Pre-treatment with (-)- deprenyl prevent the effect of neurotoxins like MPTP, 6-hydroxydopamine, ***DSP*** - ***4*** , AF64A by inhibiting the conversion of the pretoxin to toxin, or by inhibiting the neuronal reuptake mechanisms, or the combination of the two processes. However, effects of the inhibitor cannot be ruled out. (-)-Deprenyl, but not its (+)-enantiomer, proved to be a potent inhibitor of programmed cell death (apoptosis) of PC12 cells and that of ***human*** melanoma cells, in a concn. which does not induce MAO-B increases with age and the age related changes led to an overprodn. of neurotoxic agents. The inhibition of the enzyme activity can play a preventive role against neurodegenerative brain disorders. The most widely used MAO-B inhibitor in the therapy is (-)-deprenyl and it lacks the "cheese reaction". The complex mechanism for the lack of the former effect is not fully known.

L8 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2002 ACS
1996:453498 Document No. 125:132563 The analgesic activity of epicoprostanol, the major sterol of ambergis. Taha, Sadek A.; Ginawi, Omer T. (College Pharmacy, King Saud University, Riyadh, 11451, Saudi Arabia). Pak. J. Pharmacol., 12(1), 51-56 (English) 1995. CODEN: PJPHEO. ISSN: 0255-7088.
AB The analgesic activity of epicoprostanol, the major sterol of ambergis was investigated in male mice, using the hot plate method. Epicoprostanol (1, 10, 100 mg/kg i.p.) significantly increased the hot plate reaction times measured at 60 min post injector, P. chlorophenol alanini which depletes central 5-HT, only inhibited the antinociceptive activities of 1 and 10 mg epicoprostanol, whereas ***DSP*** - ***4*** , a neurotoxin of central noradrenergic the nor adrenergic and serotonergic systems may also be involved in this activity of epicoprostanol.

L8 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2002 ACS
1992:645420 Document No. 117:245420 Phencyclidine and auditory sensory gating in the hippocampus of the rat. Miller, Christine L.; Bickford, Paula C.; Luntz-Leybman, Vera; Adler, L. E.; Gerhardt, G. A.; Freedman, R. (Health Sci. Cent., Univ. Colorado, Denver, CO, 80262, USA). Neuropharmacology, 31(10), 1041-8 (English) 1992. CODEN: NEPHBW. ISSN: 0028-3908.
AB The psychotomimetic drug 1-(1-phenylcyclohexy)piperidine (PCP, phencyclidine) was found to cause a deficit in the gating of the response of the hippocampal neuron to repeated auditory stimuli, which is similar to a particular physiol. feature obsd. in ***human*** psychosis. Other drugs, with sigma agonist and/or N-methyl-D-aspartate (NMDA) antagonist effects, were administered and their ability to cause a loss of auditory gating was compared to that of PCP. The rank order of effectiveness was levoxodrol > PCP and MK-801 > N-allylnormetazocine (SKF 10047) > dexoxodrol > 3-(.+-.)2-carboxypiperazine-4-yl) propyl-1-phosphonate (CPP). Further studies of two of the drugs, PCP and MK-801, showed that selective lesioning of the noradrenergic input with the neurotoxin ***DSP4*** , as well as less selective depletion of monoamines with reserpine, blocked the loss of gating. Phencyclidine, and other drugs with the same spectrum of action, most likely disrupt gating by increasing noradrenergic activity through a sigma mechanism.

L8 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2002 ACS
1992:405422 Document No. 117:5422 The involvement of guanine nucleotide binding proteins in the pathogenesis and treatment of affective disorders. Avissar, Sofia; Schreiber, Gabriel (Health Sci. Cent., Ben-Gurion Univ. Negev, Israel). Biol. Psychiatry, 31(5), 435-59 (English) 1992. CODEN: BIPCBF. ISSN: 0006-3223.
AB It has been previously shown that lithium selectively attenuates the function of Gs proteins in the CNS. In the present work, the authors show that inhibition by lithium of muscarinic receptor-coupled G protein function is also selective to the CNS. The clin. profile of lithium, carbamazepine, and electroconvulsive treatment (ECT), agents that are effective in the prevention and treatment of bipolar affective disorder, differs from that of purely antidepressant drugs. Antidepressant drugs are effective in the acute treatment and prevention of depression only, and can even ppt. hypomanic or manic "switches," or "rapid cycling" between mania and depression. The authors investigated and compared the effects of chronic antibipolar and antidepressant treatments on receptor-coupled G protein function. Antibipolar treatments (lithium, carbamazepine, ECT) attenuate both receptor-coupled Gs and non-Gs (i.e., Gi, Go) proteins function; in contrast, only Gs protein function is inhibited by antidepressant drugs [either tricyclics or monoamine oxidase (MAO) inhibitors]. Moreover, an integral adrenergic neuronal system is required for antidepressant inhibition of Gs protein function, as pretreatment with the noradrenergic neurotoxin ***DSP*** - ***4*** (N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine) specifically abolishes the effects of antidepressant drugs on Gs protein, whereas antibipolar drug effects on G protein function are unaffected by ***DSP*** - ***4*** .

L8 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2002 ACS
1987:168977 Document No. 106:168977 Repeated electroconvulsive shock prevents increased neocortical .beta.1-adrenoceptor binding after ***DSP*** - ***4*** treatment in rats. Dooley, David J.; Heal, David

J.; Goodwin, Guy M. (Goedecke Res. Inst., Freiburg, D-7800, Fed. Rep. Ger.). Eur. J. Pharmacol., 134(3), 333-7 (English) 1987. CODEN: EJPHAZ. ISSN: 0014-2999.

AB Repeated electroconvulsive shock (ECS) was administered to rats previously injected with ***DSP*** - ***4*** (N-(2-chloroethyl)-N-ethyl-2-brmobenzylamine HCl, a noradrenergic neurotoxin. The normal increase in neocortical .beta.1-adrenoceptor binding caused by noradrenaline [51-41-2] depletion was effectively prevented by ECS. The plasticity of the .beta.1-adrenoceptor may thus be partially independent of endogenous noradrenaline concn. Addnl., functional noradrenergic neurons are not necessarily a crit. requirement for the antidepressant effect of electroconvulsive treatment in ***humans*** .

=> S SEQUENCE;S L9 AND L7
496156 SEQUENCE
355848 SEQUENCES
L9 594134 SEQUENCE
(SEQUENCE OR SEQUENCES)

L10 62 L9 AND L7

=> S L10 NOT L8
L11 60 L10 NOT L8

=> D 1,7-10,20,60 CBIB ABS

L11 ANSWER 1 OF 60 CAPLUS COPYRIGHT 2002 ACS
2002:293828 Protein and cDNA ***sequences*** of a novel ***human*** ***dual*** ***specificity*** ***phosphatase*** ***sequence*** homolog and diagnostic and therapeutic uses thereof. Weich, Nadine (Millennium Pharmaceuticals, Inc., USA). PCT Int. Appl. WO 2002031132 A2 20020418, 125 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US31661 20011010. PRIORITY: US 2000-686673 20001011.

AB The invention provides protein and cDNA ***sequences*** of a novel ***human*** , designated 8843, which has ***sequence*** homol. with ***dual*** ***specificity*** ***phosphatase*** members. The invention also provides methods of modulating the differentiation and proliferation of erythroid progenitors, and CD34 pos. cells. The invention further provides methods of treating, preventing and diagnosing erythroid-assocd. disorders such as anemias, leukemias, and erythrocytosis.

L11 ANSWER 7 OF 60 CAPLUS COPYRIGHT 2002 ACS
2001:763202 Document No. 135:314475 Protein and cDNA ***sequences*** of ***human*** ***dual*** ***specificity*** ***phosphatase*** (DUSP10) ***sequence*** homolog, and uses thereof in therapy, diagnosis, and drug screening. Duecker, Klaus (Merck Patent G.m.b.H., Germany). PCT Int. Appl. WO 2001077340 A1 20011018, 43 pp. DESIGNATED STATES: W: CA, JP, US; RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-EP3966 20010406. PRIORITY: EP 2000-107143 20000410.

AB This invention provides protein and cDNA ***sequences*** for a newly identified ***human*** protein DUSP10, which is belived to encode a novel member of ***dual*** ***specificity*** ***phosphatase*** family, since it shows homol. with HSU27193. In one embodiment, the invention relates to diagnostic assays for detecting diseases assocd. with inappropriate ***dual*** ***specificity*** ***phosphatase*** ***sequence*** homolog activity or levels. Also disclosed are methods for utilizing ***sequence*** homolog in drug screening assays and in therapy directed against diseases assocd. with inappropriate ***dual*** ***specificity*** ***phosphatase*** ***sequence*** homolog activity or levels.

L11 ANSWER 8 OF 60 CAPLUS COPYRIGHT 2002 ACS
2001:731029 Document No. 135:284077 Protein and cDNA ***sequences*** of a novel ***human*** ***dual*** ***specificity*** ***phosphatase*** ***sequence*** homologs and uses thereof. Meyers, Rachel A. (Millennium Pharmaceuticals, Inc., USA). PCT Int. Appl. WO 2001073060 A2 20011004, 138 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US9603 20010322. PRIORITY: US 2000-PV191858 20000324.

AB The invention provides protein and cDNA ***sequences*** of a novel ***human*** protein, designated 18221, which has ***sequence*** homol. with ***dual*** ***specificity*** ***phosphatase*** family members. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. 18221 nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 18221 gene has been introduced or disrupted. The invention still further provides isolated 18221 proteins, fusion proteins, antigenic peptides and anti-18221 antibodies. Diagnostic methods utilizing compns. of the invention are also provided. The invention also provides methods of modulating the differentiation and proliferation of hematopoietic cells (e.g., erythroid cells) utilizing the compns. of the invention. Accordingly, methods of treating, preventing and/or diagnosing hematopoietic disorders are disclosed.

L11 ANSWER 9 OF 60 CAPLUS COPYRIGHT 2002 ACS
2001:731028 Document No. 135:284076 Protein and cDNA ***sequences*** of novel ***human*** ***dual*** ***specificity*** ***phosphatase*** ***sequence*** homologs and uses thereof. Meyers, Rachel A. (Millennium Pharmaceuticals, Inc., USA). PCT Int. Appl. WO 2001073059 A2 20011004, 143 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US9477 20010323. PRIORITY: US 2000-PV191858 20000324.

AB The invention provides protein and cDNA ***sequences*** of ***human*** proteins, designated 38692 or 21117, which have ***sequence*** homol. with ***dual*** ***specificity*** ***phosphatase*** family members. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. 38692 or 21117 nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 38692 or 21117 gene has been introduced or disrupted. The invention still further provides isolated 38692 or 21117 proteins, fusion proteins, antigenic peptides and anti-38692 or 21117 antibodies. Diagnostic methods utilizing compns. of the invention are also provided.

L11 ANSWER 10 OF 60 CAPLUS COPYRIGHT 2002 ACS
2001:661622 Document No. 135:223454 Protein and cDNA ***sequences*** of a novel ***human*** ***dual*** ***specificity*** ***phosphatase*** and uses thereof. Kapeller-Libermann, Rosana (Millennium Pharmaceuticals, Inc., USA). PCT Int. Appl. WO 2001064911 A2 20010907, 134 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2.

APPLICATION: WO 2001-US6177 20010227. PRIORITY: US 2000-PV185772 20000229; US 2000-704139 20001101.

AB The invention provides protein and cDNA ***sequences*** of a novel ***human*** protein, designated 18232, which is a novel member of ***dual*** ***specificity*** ***phosphatase*** family. The invention also provides antisense nucleic acid mols., recombinant expression vectors contg. 18232 nucleic acid mols., host cells into which the expression vectors have been introduced, and nonhuman transgenic animals in which a 18232 gene has been introduced or disrupted. The invention still further provides isolated 18232 proteins, fusion proteins, antigenic peptides and anti-18232 antibodies. Diagnostic methods utilizing compns. of the invention are also provided. The invention also provides methods of modulating the differentiation and proliferation of hematopoietic cells (e.g., erythroid cells) utilizing the compns. of the invention. Accordingly, methods of treating, preventing and/or diagnosing erythroid-assocd. disorders such as anemias, leukemias, and erythrocytosis are disclosed. Tissues in which the ***dual*** ***specificity*** ***phosphatase*** 18232 gene is highly expressed include fetal liver, kidney, lung, skeletal muscle, CD8 pos. cells, bone marrow, blood cells and epithelial cells. Hence, the ***dual*** ***specificity*** ***phosphatase*** is relevant to disorders involving the tissues in which it is expressed.

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2001:137383 Document No. 134:189007 Protein and cDNA ***sequences*** of novel ***human*** and mouse protein phosphatases and uses there of in diagnosis and treatment of phosphatase-related disorders. Plowman, Gregory D.; Martinez, Ricardo; Whyte, David; Hill, Ron; Flanagan, Peter; Lioubin, Mario (Sugen, Inc., USA). PCT Int. Appl. WO 2001012819 A2 20010222, 138 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US22158 20000811. PRIORITY: US 1999-PV149005 19990813.

AB The present invention relates to novel mammalian protein phosphatase polypeptides, nucleotide ***sequences*** encoding the novel kinase polypeptides, as well as various products and methods useful for the diagnosis and treatment of various protein phosphatases-related diseases and conditions.. Preferably, the polypeptides of the present invention belong to the dual-specificity group of protein phosphatases. Through the use of a "motif extn." bioinformatics script, addnl. ***human*** and mouse members of the phosphatase family are herein presented. These phosphatases include MKP-like proteins, a CDC14-like protein, a PTEN-like protein, and myotubularin (MTM)-like proteins. Classification of proteins as new members of established families has proven highly accurate not only in predicting motifs present in the remaining non-catalytic portion of each protein, but also in their regulation, substrates, and signaling pathways. The cDNA clones encoding novel ***human*** and mouse protein phosphatases were isolated by searching for signature ***sequences*** in the public EST databases. ***Dual*** ***specificity*** ***phosphatases*** were identified using an HMM model built from DSPs from mammalian and non-mammalian sources. ESTs were translated in six open reading frames and were searched against the models. The public EST database was also searched by BLAST with representative members of the various families, such as ***human*** DUS6, ***human*** MTM1, and ***human*** PTEN1. Full-length ***sequence*** extension of the EST clones is achieved using cDNA and genomic databases (e.g., Celera and ***Human*** Genome Program databases). Domain and motif identification, chromosomal location, and tissue expression distribution are also provided for the protein phosphatases genes.

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1993:489966 Document No. 119:89966 Expression cloning of a ***human*** ***dual*** - ***specificity*** ***phosphatase*** . Ishibashi, Toshio; Bottaro, Donald P.; Chan, Andrew; Miki, Toru; Aaronson, Stuart A. (Lab. Cell. Mol. Biol., Natl. Cancer Inst., Bethesda, MD, 20892, USA).

Contrary to expectation, this broadly expressed enzyme did not inactivate MAPKs in transient co-transfection assays but instead displayed the capacity to function as a selective activator of the MAPK Jnk, hence the name, Jnk Stimulatory Phosphatase-1 (JSP-1). This study illustrates a new aspect of the regulation of MAPK-dependent signal transduction and raises the possibility that JSP-1 may offer a different perspective to the study of various inflammatory and proliferative disorders assocd. with dysfunctional Jnk signaling.

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2000:646042 Document No. 133:236826 DSP-1 ***dual*** - ***specificity*** ***phosphatase*** . ***Luche, Ralf M.*** ; ***Wei, Bo*** (Ceptyr, Inc., USA). PCT Int. Appl. WO 2000053636 A2 20000914, 74 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US6154 20000308. PRIORITY: US 1999-PV123255 19990308.

AB Compns. and methods are provided for the treatment of conditions assocd. with cell proliferation, cell differentiation and/or cell survival. In particular, the ***dual*** - ***specificity*** ***phosphatase*** DSP-1, and polypeptide variants thereof that stimulate dephosphorylation of DSP-1 substrates, are provided. The polypeptides may be used, for example, to identify antibodies and other agents that inhibit DSP-1 activity. The polypeptides and agents may be used to modulate cell proliferation, cell differentiation and cell survival for such disorders include cancer, graft-vs-host disease, autoimmune disease, allergies, metabolic disease, and abnormal cell growth or proliferation, and cell cycle abnormalities.

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